

## INHALATION INJURY AND TOXIC INDUSTRIAL CHEMICAL EXPOSURE

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Supersedes:	Inhalation Injury and Toxic Industrial Chemicals (TICs), updated Apr 2008		

**1. Goal.** There are multiple toxic industrial chemicals (TICs) that act on the respiratory tract. This CPG reviews the four most common and toxic TICs. More information is available from the CDC at <http://www.bt.cdc.gov/agent/agentlistchem-category.asp> and in the *Textbook of Military Medicine*,

[http://www.bordeninstitute.army.mil/published\\_volumes/biological\\_warfare/biological.html](http://www.bordeninstitute.army.mil/published_volumes/biological_warfare/biological.html)

**2. Background.** In general, the treatment of Acute Lung Injury (ALI) and Acute Respirator Distress Syndrome (ARDS), secondary to TICs is similar to that for smoke inhalation injury: i.e., largely supportive with a focus on (1) airway management, (2) lung-protective ventilation strategies, (3) aggressive pulmonary toilet, and (4) avoidance of volume overload or rapid fluid infusion that might worsen pulmonary edema secondary to capillary leak. All patients requiring mechanical ventilation (MV) secondary to TIC inhalation are at a higher risk of developing ventilator-associated pneumonia and should be monitored closely. The treatments mentioned below are largely based on animal experiments, and evidence for clinical use in humans is limited. All specific drug regimens are most likely to work best if implemented immediately after exposure and inhalation. Finally, patients requiring MV should be transferred to a burn center with expertise in inhalation injury, if feasible.

- a. Chlorine (Cl<sub>2</sub>). Used abundantly in industry; it is a common cause of industrial and transportation accidents and may be used in military weapons such as IEDs. Dissolves in water to form HCl and HOCl acids; all 3 types participate in pathogenicity and causes both small airway and alveolar injuries. Immediate effects of Chlorine gas exposure are inflammation of the upper respiratory tract, cough, shortness of breath, chest pain, choking, and headache. Proposed treatments include inhaled (nebulized) corticosteroids (e.g. budesonide), nebulized sodium bicarbonate in water (e.g. 3.75 to 4.2%), nebulized beta agonists (e.g. terbutaline), and prone position MV.<sup>1</sup> (See Appendix A.)
- b. Phosgene (COCl<sub>2</sub>). Smells like newly mowed hay. Used in production of plastics, drugs, and polyurethane. Most lethal chemical agent during WWI. Classic presentation is that of delayed-onset pulmonary edema that is triggered by exertion. Pathophysiology includes oxidative stress and influx of neutrophils into the lung. Proposed treatments focus on these mechanisms, and include N-Acetylcysteine, ibuprofen, aminophylline, isoproterenol, and colchicine.<sup>2</sup>
- c. Hydrogen sulfide (H<sub>2</sub>S). Smells like rotten eggs. Seen in petroleum, waste management (“dung lung”), and the natural gas industries. Enters the bloodstream via the lungs, binds to Cytochrome c oxidase (similar to cyanide); prevents oxygen utilization by the mitochondria.

The phenomenon of “knockdown,” or sudden loss of consciousness due to its effects on brainstem mitochondria, is common. Other effects are seizures and myocardial ischemia. Direct toxic effect on lungs causes pulmonary edema. Direct effect on cornea causes

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keratoconjunctivitis (“gas eye”). Treatment may include i.v. Nitrites, which must be used carefully with cyanide poisoning to avoid excessive levels of methemoglobinemia.<sup>3</sup>

- d. Anhydrous ammonia (NH<sub>3</sub>). Found in fertilizer, refrigeration, petroleum, and explosives industries. Transported in liquid form at sub-zero temperatures. Reacts quickly upon release to form NH<sub>4</sub> (ammonium hydroxide) – a strong base. Causes alkali skin burns and frostbite. May cause rapid, and severe upper airway obstruction if inhaled, followed by pulmonary edema. Inhaled corticosteroids were not effective in animal models, suggesting the directly destructive mechanism of this chemical. Patients with skin and ocular involvement must be copiously irrigated with water, as with any alkali injury.

**3. Responsibilities.** It is the trauma team leader’s responsibility to ensure CPG adherence.

### **4. References.**

<sup>1</sup> Wang J, et al. Administration of aerosolized terbutaline and budesonide reduces chlorine gas-induced acute lung injury. *Journal of Trauma*. Apr 2004;56(4):850-62

<sup>2</sup> Sciuto AM, Hurt HH. Therapeutic treatments of phosgene-induced lung injury. *Inhalation Toxicology*. 16(8):565-80, Jul 2004.

<sup>3</sup> van Aalst JA et al. Hydrogen sulfide inhalation injury. *Journal of Burn Care & Rehabilitation*, 21(3):248-53, May-Jun 2000

Approved by CENTCOM JTTS Director, JTS Director  
and Deputy Director and CENTCOM SG

## APPENDIX A CHLORINE INHALATION

### FIELD:

- Check for co-contamination → decontaminate if needed
- Assess ABCs
- Mask O<sub>2</sub>/intubation if needed to maintain SpO<sub>2</sub>>90%
- Flush eyes/nose/mouth with water
- Bicarb/NS neb if available (2cc ACLS 8.5% bicarb ampoule: 2cc NS) x 1
- Albuterol neb/inhaler for wheezing

### EMT:

- Receive patient; check for co-contamination → decontaminate if needed
- Reassess ABCs and secondary survey
- High flow humidified O<sub>2</sub> (if not intubated)
- Fluorescein/slit lamp exam to rule out corneal burns
- Irrigate eyes/nose with saline; erythromycin ointment to eyes
- Bicarb/NS neb (2cc ACLS 8.5% bicarb ampoule: 2cc NS) x 1 (if not already done)
- Albuterol nebs (can add ipratropium for improved bronchodilation) q2h and prn for wheezing
- Obtain CXR, draw blood for rainbow labs
- Can discharge safely if normal CXR and no wheezing or O<sub>2</sub> requirement >6 hrs post exposure
- Admit to ward/ICU otherwise

### ICU:

- Receive patient, reassess ABCs
- Place arterial line (also CVC if needed)
- RT care
  - High flow humidified O<sub>2</sub> (if not intubated)
  - Duonebs (albuterol/ipratropium) q2h & prn
  - Flovent (fluticasone 220mcg) 2 puffs immediately then bid
  - Consider short course high dose IV steroids for airflow obstruction (wheezing, etc.)

### POST-DISCHARGE/LONG TERM FOLLOW-UP:

- CXR 1 month post exposure—look for persistent infiltrates or volume loss
- Consider formal pulmonary function testing with lung volumes and DLCO to assess for scarring
- For abnormal PFTs, consider high-resolution CT scanning to assess for pulmonary fibrosis

### NOTES:

- Chlorine patients may have considerable laryngeal edema; anticipate difficult airway when intubating.
- Patients will need aggressive pulmonary toilet to remove necrotic tissue as their inhalation injury matures. Blocked endotracheal tubes (ETTs) are common, especially in children with

small tubes. Frequent bronchoscopy may be needed in addition to suctioning to keep ETT patent.

- Patients are at high risk for developing secondary pneumonia due to mucosal injury and loss of normal lung defenses. Use low threshold for starting antibiotics if fever, elevated WBC count, and/or purulent secretions develop.
- Acute Respiratory Distress Syndrome (ARDS)
  - High airway pressures will be needed to maintain oxygenation. Patients require daily exam and CXR to look for barotrauma (subcutaneous air, pneumothorax) and need for chest tube.
  - Be mindful that suctioning patients with ARDS can cause immediate and profound O<sub>2</sub> desaturations. This risk must be balanced against the benefit of keeping the ETT unblocked.
  - Patients may require high dose sedation to maintain synchrony with ventilator. The unavoidable consequence of these agents is prolonged weaning (1-2 weeks).
  - ARDS patients will experience O<sub>2</sub> desaturations with agitation. They may require intermittent paralysis with neuromuscular blocking agents in addition to sedation to maintain oxygenation.
  - Patients may need to be placed in prone position if difficult to oxygenate by any other means.
  - Patients experience frequent episodes of decompensation. Possibilities include dislodged ETT, blocked ETT, break in vent circuit, agitation, aggressive suctioning, pneumothorax.

### CLINICAL EFFECTS OF CHLORINE EXPOSURE

#### Immediate Effects of Chlorine Gas Toxicity

- |   |   |
|---|---|
| • Inflammation of the conjunctivae, nose, pharynx, larynx, trachea, and bronchi | • Dizziness   |
| • Local airway edema  | • Muscle weakness                                       |
| • Cough (52-80%)  | • Ocular and nasal irritation (4-6%)                    |
| • Shortness of breath (20-51%)  | • Burning sensation in throat and substernal area (14%) |
| • Chest pain (33%)  | • Nausea and/or vomiting (8%)                           |
| • Choking   | • Abdominal discomfort                                  |
| • Headache  |   |

*Later* effects of chlorine gas toxicity include pulmonary congestion due to plasma exudation that fills the alveoli with fluid. The *hallmark* of pulmonary injury associated with chlorine toxicity is pulmonary edema, manifested as hypoxia.

#### Background

- Chlorine gas is a pulmonary irritant with intermediate water solubility that causes acute damage in the upper and lower respiratory tract.
- Chlorine gas was first used as a chemical weapon at Ypres, France, in 1915. Of the 70,552 American soldiers poisoned with various gases in World War I, 1843 were exposed to chlorine gas.

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- Internationally and in the U.S., chlorine gas accounts for the largest single cause of major toxic release incidents.
- Chlorine gas has been used as weapon in the Iraq Theater of Operations. Suicide truck bombs, chlorine bombs, and IEDs containing chlorine have exploded, exposing military personnel and civilians to chlorine gas.

### **Pathophysiology**

- Chlorine is a greenish-yellow, noncombustible gas at room temperature and atmospheric pressure.
- Chlorine is more dense than air, causing it to remain near ground level and increasing exposure time.
- Exposure to chlorine gas may be prolonged because its moderate water solubility may not cause upper airway symptoms for several minutes.
- The odor threshold for chlorine is approximately 0.3-0.5 parts per million (ppm); however, distinguishing toxic air levels from permissible air levels may be difficult until irritative symptoms are present.
- Chlorine reacts with water to form hypochlorous (HOCl) and hydrochloric (HCl) acids. Elemental chlorine and its derivatives may cause biological injury. Hydrochloric acid is highly soluble in water. The predominant targets of the acid are the epithelia of the ocular conjunctivae and upper respiratory mucus membranes.

*These Recommendations for Empiric Treatment are Based on the Recent Experience of the 28th CSH and Supplemented By Literature Review.*